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MUCOCELE OF THE FRONTAL SINUS.

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Retained secretions in the frontal sinus give rise to such well-marked clinical phenomena that the diagnosis of this condition is usually a matter of no great difficulty; nevertheless these cases are quite frequently mistaken by the inexperienced in ophthalmic surgery for other and more serious forms of disease. The case described in the following notes was sent to me with the diagnosis: "Tumor of the orbit, probably malignant." This was the verdict of several practitioners who had watched the case from the beginning and made many examinations with a view to discover the nature of the disease.

The patient, Mrs. M., æt. 67, widow of a physician, had been subject to nasal catarrh for thirty or forty years, and some twenty years ago was said to have polypus in the left nostril. For the removal of this she underwent several rather severe operations, and states that some powerful caustics were used, in addition to surgical instruments. Probably the course of treatment will account for the condition of the left nostril, when she came under my notice. About two-thirds of an inch from the orifice a firm cuticular mass stretched across the lumen of the nasal cavity and formed a complete obstruction, excepting a small aperture about

one and one-half lines in diameter, close to the floor of the nostril. Through this a weak current of air and a small amount of nasal secretion could be forced by a strong effort of expiration. The voice was thick and nasal, a peculiarity which had existed for many years.

The mucous membrane of the right nostril was generally thickened, and the nasal secretions somewhat in excess, but no polypi discoverable, though she at times experienced almost complete closure of this nostril also, and is at all times "a mouth-breather."

I may here state that of the five cases of disease of the frontal sinus, which I have seen in the past four years, this one was the third in which polypi of the nostrils were or had been present. In one case both nostrils were completely filled with numerous polypoid masses, which I removed at the time of operating for the cure of a fistula through the upper eye-lid.

The connection between nasal polypi and disease of the frontal sinus, which these cases seem to suggest, is, however, of too obvious a nature to require more than a passing mention. Beyond a certain degree of anxiety and depression of spirits, due to the unfavorable diagnosis and prognosis already alluded to, patient had nothing to complain of, though her breathing at night often caused great annoyance to her friends who happened to be within hearing distance.

Certainly her general health had not suffered from the disease, nor had she suffered actual pain in or about the diseased orbit—nothing more in fact than an unpleasant feeling of fullness which became almost painful when stooping or lowering the head forward.

About a year previously she had first noticed an increased fullness of the left eye, and this had gradually augmented to the condition shown by figure 4.[1] Roughly estimated, the displacement of the eye-ball was one and one-half inch downward, one-half inch forward, and one-fourth inch outward. The movements of the eye-ball, though restricted, especially in an upward

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1. The drawings, 4 and 5, were made by the editor from the original photographs furnished by Dr. Buller.

from the normal ophthalmoscopic appearance of the fundus oculi.

A large, ill-defined, elastic, fluctuating mass occupied the inner and upper portion of the orbit. A formation presenting these characteristics and in this situation, of course, at once suggests disease of the frontal sinus. To establish the diagnosis a small trocar and canula was plunged into the most prominent part of and inward direction, were by no means lost, nor was the position of the upper lid such as to deprive the eye of its natural protection. Vision was normal, as might have been inferred

FIG. 4.

the swelling, and when withdrawing the trocar and pressing moderately upon the surrounding parts, a thick, dark grey viscid mucus was forced out of the canula, and at the same time, by using the instrument as a probe, it was found to pass upwards and inward further than could possibly have been the case had the walls of the orbit been intact.

The subsequent method of treatment was in accordance with the recognized principle of draining off the frontal sinus through the nostril. On making a free incision into the swelling an enormous quantity of dark viscid mucus escaped, and an exploration of the inner wall of the orbit with the finger discovered a

very large gap in the inner wall of the orbit, with an extensive cavity beyond, lined with a thick soft mucous membrane. This cavity included the enlarged frontal sinus, as well as a space corresponding to fully half the (anterior) ethmoidal cell. Through the hiatus in the inner wall of the orbit the mucous secretion had forced its way into the orbit, not confined within cyst walls, but as an infiltration into the tissues behind and internal to the eyeball, causing displacement of the latter to the extent and in the manner described. Upon washing out the cavity and pressing the eye gently backward, it resumed nearly a normal position. A

FIG. 5.

free crucial incision was made through the obstruction in the nostril, and a perforation through its roof into the sinus, in the usual way. A perforated rubber drainage tube was passed from below through the aperture on the floor of the sinus, and out through the orbital incision, and a compression bandage applied.

The following day the eye-lids were swollen and tender. There was also nearly the same degree of proptosis as before the operation. After syringing through the tube with a 10 per

cent. solution of boracic acid, lead lotion was applied to the lids instead of the compressive bandage. Under this treatment the inflammatory action gradually subsided, and the patient returned to her home in the country with directions to wear the tube for several weeks, and keep the parts clean by syringing with the same solution. These directions appear to have been imperfectly carried out, inasmuch as she removed the tube a week after she returned home, and allowed the external wound to heal up.

Some eighteen weeks after the operation, as seen by figure 5, the displacement of the eye-ball was still quite appreciable, but was thought to be gradually diminishing. A re-accumulation of the mucous secretion does not seem an impossible contingency, and although no great harm is likely to ensue, should this occur, it is to be regretted the drainage tube was not retained for a considerably longer period. Viewed from the light of my own experience in disease of the frontal sinus, there are several features in this case which seem unusual. First, there was the absence of all inflammatory action during the whole course of the disease; secondly, the accumulation of mucus was unusually large; thirdly, the erosion or absorption of bone which permitted infiltration of the orbit with mucus was much greater than I have ever seen from this cause. Long continued pressure was obviously the cause of this absorption, and if the anterior ethmoidal cells were crowded with the same secretion, the pressure must necessarily have been exercised over an unusually large area, and absorption may have been far advanced over this area before the eye-ball became displaced or the patient aware of her disease.

A CASE OF SARCOMATOUS TUMOR OF THE ORBIT.

DR. H. H. VINKE, M. D., ST. CHARLES.

I desire to report the following case, as it serves to show the benefit to be derived from the extirpation of malignant tumors of the orbit, when the same is complete.

About five months ago I was called upon by my friend, Dr. C. M. Johnson, to see with him A. J., a colored woman of 42, who had been suffering excessive pains for months in the left eye. Upon examination I found considerable exophthalmus; the movements of the eye-ball were abolished. The upper lid was œdematous, and the lower one greatly everted, its conjunctiva being swollen and fleshy in appearance. Vision was completely lost. General condition of patient was poor, pulse small and nearly 100. We diagnosticated a tumor in the orbit, behind the eye, causing the eye-ball to protrude and destroying vision, and decided to operate upon her the next day. The next day, therefore, with the assistance of Drs. Johnson and Morgner, I extirpated the eye-ball and removed, as I thought, the tumor entirely. This tumor weighed about ℥v, and evidently sprang from the connective tissue of the orbit. We advised the orbital cavity to be washed out with carbolized water daily, and ordered carbolized water dressings. The tumor, however, rapidly recurred. About a month after the operation I was called to see her again, as she was again suffering considerable pain. I found a large tumor protruding between the eye-lids, filling the entire orbital cavity, and infiltrating the upper and lower eye-lids, especially the lower one, to a considerable extent. I decided to operate upon her a second time. Again being assisted by the above gentlemen, I began the operation by dividing the outer canthus, then making an incision one inch upwards and another two inches downwards, both of these incisions meeting the other at right angles. After dissecting up the lower and upper eye-lids, and turning up the flaps, the tumor was completely exposed, and it was found to be an immovable growth and widely attached to the periosteum of the orbit. The tumor had to be removed piecemeal, and after all that could be gouged out with the scalpel, curved scissors and handle of scalpel had been removed, the orbit was carefully scraped out with a curette, so that the orbit was altogether denuded of its periosteum.

After this the infiltrated portions of the eye-lids were carefully dissected off, so that no remnant of the tumor was left behind. The flaps were now turned back to their original place and stitched.

There was some hemorrhage, which however was readily

checked by the thermo-cautery. The orbital cavity was washed out with carbolizad water, and pieces of lint, spread with the chloride of zinc paste, were applied to bottom and walls of the orbit. After both operations there was little or no fever; the pain was always immediately removed after the operations. There was a considerable amount of pus discharged from the orbit for weeks after the second operation, on which account she was directed to syringe out the cavity twice a day with carbolized water. The constitutional treatment consisted of tonics, quinine, iron, etc. I will state here that the tumor, which was removed at the second operation, weighed 3½ ozs. Although four months have passed since the last operation, there has been no sign of recurrence, and I do not apprehend that the tumor will recur, as she is now in the enjoyment of perfect health. I am inbebted to Dr. O. Morgner for examining the tumor microscopically, and determining that it was a fibro-sarcoma, the recurrent fibroid of Sir J. Paget.

INJURY. OTITIS MEDIA PURULENTA. RIGHT-SIDED HEMIANOPIA. AUTOPSY. ABSCESS NEAR THE GYRUS ANGULARIS IN LEFT OCCIPITAL LOBE OF THE BRAIN.

BY ADOLF ALT, M. D.

The following case is of especial interest, on account of its bearing upon the theory of cerebral localization. Authentic cases of this kind are not frequently seen, and have thus far been reported in so small a number, that I think it well to add each single case to the series, as it is observed.

L. S. æt. 49, a robust, healthy looking gentleman came to consult me about his left ear on August 29. 1881. He stated that he had frequently in former years, and especially of

late, suffered from malaria. Within the last two months this had shown itself chiefly in the form of neuralgia in the left side of his head. In consequence of the considerable pain he had been very sleepless. A few days before consulting me he had, as he thought, caught cold in his left ear and accordingly the pain had become yet more aggravated. Quite accidentally he stated also that three months previous he had received a severe contusion on the left side of the head behind the ear, being struck by the heavy curtain-pole of a summer street-car while passing it in another one.

Status praesens.—The left auricle as well as the walls of the external auditory meatus are considerably swollen. No view of the membrana tympani can be had. There is an œdematous swelling over the mastoid. h=o. No bone conduction.

I made an incision through skin and periosteum on the mastoid, but found the bone healthy. Then I made two incisions into the swollen tissue of the external auditory meatus. The patient was then ordered to steam the ear. By this treatment he felt greatly relieved, the swelling disappeared in a few days and a profuse discharge from the middle ear set in. Inspection showed now a perforation of the membrana tympani in the lower posterior quadrant.

When under proper treatment, the discharge ceased, the general condition of the patient became again worse. When by steaming the ear the discharge was brought on again, he felt better. Trephining of the mastoid bone was refused. Thus the case went on for about three weeks alternately improving and getting worse. During this time the patient complained sometimes of dizziness, he could walk but slowly, and his memory became affected in such a way, that he "forgot even words" and had often to stop in the middle of a sentence, not being able to find the needed words. There also appeared a small teat-like granulation on the upper wall of the external meatus near the outer orifice. When pressure was exerted on this little tumor a minute central opening at its apex discharged a drop of pus. A probe which I introduced into this opening slid easily in, in an upward, backward and inward direction, without encountering any obstacle and to such a distance, that I

stopped, being afraid of breaking through the dura mater. He again refused the opening of the mastoid.

A few days after this exploration, he complained of loss of sight. An ophthalmoscopic examination revealed no pathological changes in the back-ground of the eyes. The field of vision was, however, greatly restricted for the right side of each eye and in a few days more this condition changed into a complete right-sided hemianopia.

The patient's general condition together with this symptom pointed now to the diagnosis of cerebral abscess in the left hemisphere. Whether this was caused by the injury or by the ear-affection remained undecided.

I did not see the patient again until invited to the autopsy on the 17th of October.

The autopsy (made by Dr. G. Richter with the assistance of Dr. O. Greiner) was obtained with great difficulty, and we were continually interrupted.

We found a large cavity, filled with pus, lying between the dura mater and the cranium just at the site of the injury, and corresponding to it an abscess of the size of an English walnut, barely covered with a very thin coat of cortex, in the substance of the left occipital lobe of the brain and close to the gyrus angularis. No communication apparently existed between the ear and the intracranial pus-cavities.

GLAUCOMA SIMPLEX IN A HYPERMETROPIC EYE AFTER TENOTOMY.

BY J. S. PROUT, M. D., BROOKLYN, NEW YORK.

A man, æt. 54, consulted me June 20. 1872. The right eye converged ordinarily but he could use either eye, at will, for near or distant objects. He wore $+\frac{1}{9}$ for reading and, when not doing so, the eye not in use converged about three lines; when looking at distant objects the right eye converged about $1\frac{1}{2}$ lines. O. D. V. $=\frac{8}{100}$; with $+\frac{1}{20}$ spher. $=\frac{20}{30}$. O. S. V $=\frac{20}{64}$, with

$+\frac{1}{20}=\frac{20}{30}$. He said he wished to be able to look people straight in the face, so I agreed to divide the internal rectus O. D., and did so freely four days later, without ænesthetic. As there was good converging power I put in an abducting suture, which, six hours later, was removed on account of pain, when the ball was found to be in good position with very little converging power. Five days after the operation there was a slight tendency to converge. He was strongly urged to wear correcting glasses ($\frac{1}{20}$ spher.)but refused on the ground that his employers would think, if he did so, that he was losing his sight and would dismiss him. Twenty-four days after the operation, as there was more convergence, he seemed half disposed to try the correcting glasses, but did not do so.

1873. January 6.—He said that for two weeks there had been some cloudiness of vision in O. D. External appearance normal, O. D. T. +1? O. S. T. n. V. O. D.$=\frac{12}{200}$, with $+\frac{1}{20}=\frac{20}{40}$; O. S, $\frac{20}{80}$ with $\frac{1}{20}=\frac{20}{40}$. The ophthalmoscope showed media clear. O. D. disc pearl gray color, cupped physiologically, could not obtain pulsation by moderate pressure. No changes recognized at macula, striped pigmentation in other parts of the fundus. O. S. nearly same general appearances—nerve disc more natural color.

April 14.—O. D. visual field limited up and in, disc pale; moderate pressure caused pulsation.

1875. June 22.—O. D. T.+1, upper, inner quadrant of visual field nearly lost, limited in other directions. Disc cupped, arterial pulsations easily produced. O. S. T. n. Visual field normal.

1876. January 8.—Yesterday he had a temporary loss of V. O. D. Now V. O. D. with $+\frac{1}{20}=\frac{16}{50}$; O. S. V. with$+\frac{1}{20}=\frac{16}{20}$. O. D. T.+2 Nerve disc deeply cupped, atrophic ring (halo) round disc. Advised iridectomy. Refused.

1878. Feb. 27.—O. D. T.+? Eyes externally normal in appearance, has had no pain. O. D. can only see large objects dimly. O. S. T. n., Visual field normal.

March 3—19. Made several instillations of a solution of sulphate of eserine (1 per cent), but without benefit.

1884. March 28.—Both eyes are normal in appearance. O. D.

is sightless. V. O. S. as good as before—not able to make a careful examination. The right eye has at no time been painful nor have there been phosphenes or iridescences.

In view of the fact that the left eye is still good may we assume that the operation had any influence in causing the development of the glaucoma simplex in the right, and equally hypermetropic eye?

A CASE OF RUPTURED ZONULA. LENS CONTINUING TRANSPARENT AFTER THREE YEARS. MYDRIASIS AND LOSS OF ACCOMMODATION. INCREASE OF REFRACTION UNDER INFLUENCE OF MYOTICS.

BY DR. JOHN GREEN, ST. LOUIS.

J. A. J., a farmer, 31 years of age, was first seen by me January 5. 1882. Three years before he had received an injury to the right eye from a blow by a stick of fire-wood upon or in the immediate neighborhood of the organ.

Almost immediately after the injury it was noticed that the right pupil was of about twice the diameter of its fellow, and that it was no longer possible to read moderately fine print with this eye. The dilatation of the pupil and loss of accommodative power in this eye had continued sensibly unchanged.

The pupil presented an apparent diameter of between six and seven millimetres, and did not contract under the influence of strong light or in the act of convergence and accommodation

(with the fellow eye) for the near. V. $=\frac{16}{LXXX}$, which was raised to $\frac{16}{XXXII}$ by — 1. d. sph. (O. S., V. $=\frac{16}{XXV}$): dark rainy day.

Ophthalmoscopic examination revealed a detachment of the lens from the zonula at its upper outer border, with a slight displacement of the lens downwards and inwards. About one-fourth of the free border of the lens was visible through the dilated pupil. The lens appeared perfectly transparent.

A small drop ($\frac{1}{4}$ minim) of a solution of eserine sulphate ($\frac{1}{240}$) contracted the pupil to a diameter of about 2 mm. in the course of about half an hour. At the end of an hour, M. $=\frac{1}{15}$, V. $>\frac{16}{XX}$, V. $<\frac{16}{XVI}$: sky lighter than at time of first measurement.

January 6.—Diameter of pupil a trifle less than when first seen the day before. M. $=\frac{1}{24}$, V. $=\frac{16}{XX}$, (O. S., Hm. $=\frac{1}{84}$, V. $=\frac{16}{XVI}$.)

A drop ($\frac{1}{2}$ minim) of a solution of pilocarpine hydrochlorate ($\frac{1}{240}$) was instilled, and twice again at intervals of half an hour. At the end of an hour and a half after the first instillation, the pupil measured about 3 mm. in diameter: M. $=\frac{1}{14}$, V $=\frac{16}{XX}$.

A solution of pilocarpine hydrochlorate ($\frac{1}{240}$) was ordered to be instilled in the evening and again early the next morning.

January 7. 11 A. M.—Pupil dilated about as the day before: M. $=\frac{1}{27}$, V. $=\frac{16}{XX}$. The pilocarpine solution was again instilled twice, with an interval of half an hour. An hour and a half after the first instillation the pupil was well contracted.

With $-\frac{1}{27}$ V. $=\frac{16}{CLX}$,
" $-\frac{1}{11}$ V. $=\frac{16}{XX}$,
" $-\frac{1}{10}$ V. $=\frac{16}{XVI}$,
" $-\frac{1}{9}$ V. $=\frac{16}{XX}$,

Half an hour later, M. $=\frac{1}{11}$, V. $=\frac{16}{XVI}$. (O. S. Hm. $=\frac{1}{72}$, V. $=\frac{16}{XVI}$).

January 8.—Diameter of pupil=5 mm. (O. S., diameter of pupil=3 mm).

A small drop ($\frac{1}{4}$ minim) of a solution of eserine sulphate ($\frac{1}{240}$) was instilled. The measurements made before and one hour after the instillation were as follows :

Before instillation of eserine sulphate.	One hour after instillation of eserine sulphate (gr. $\frac{1}{240}$).
Diameter of Pupil=5 mm.	Diameter of pupil=3 mm.
With $\frac{1}{\infty}$ V.=$\frac{16}{LXXX}$,	With $\frac{1}{\infty}$ V.=$\frac{16}{CLX}$,
" $-\frac{1}{36}$ V.=$\frac{16}{XXXII}$,	- - - - -
" $-\frac{1}{30}$ V.=$\frac{16}{XXV}$,	- - - - -
" $-\frac{1}{24}$ V.=$\frac{16}{X X}$,	With $-\frac{1}{24}$ V.=$\frac{16}{LXIV}$,
" $-\frac{1}{20}$ V.=$\frac{16}{X X}$,	- - - - -
" $-\frac{1}{18}$ V.=$\frac{16}{XXV}$.	- - - - -
- - - - -	With $-\frac{1}{14}$ V.=$\frac{16}{X X}$,
- - - - -	" $-\frac{1}{13}$ V.=$\frac{16}{XVI}$,
- - - - -	" $-\frac{1}{12}$ V.=$\frac{16}{XVI}$,
- - - - -	" $-\frac{1}{11}$ V.=$\frac{16}{XX}$.
M. $>\frac{1}{24}$, $<\frac{1}{20}$; V. =$\frac{16}{X X}$.	M. $>\frac{1}{13}$, $<\frac{1}{12}$; V.=$\frac{16}{XVI}$.

Increase in R. under influence of eserine=say $\frac{1}{13}-\frac{1}{24}$, $=\frac{1}{28}$; or $\frac{1}{12}-\frac{1}{20}$, $=\frac{1}{30}$.

January 9.—A solution of pilocarpine hydrochlorate (gr. $\frac{1}{240}$) had been instilled three times during the morning, the last time two hours before the (noon) visit. Diameter of pupil=5 mm., M.=$\frac{1}{20}$, V.=$\frac{16}{X X}$. (Rather poor light.)

A small drop($\frac{1}{4}$ minim)of a solution of eserine sulphate (gr. $\frac{1}{240}$) was instilled, and V. and R. measured after the lapse of an hour; M.=$\frac{1}{15}$, V. =$\frac{16}{XVI}$.

An hour later, or two hours after the eserine instillation, M.=$\frac{1}{12}$, V.=$\frac{16}{XVI}$.

January 10.—A solution of pilocarpine hydrochlorate (gr.$\frac{1}{240}$) had been instilled three times before the visit, and a stronger solution (gr.$\frac{1}{120}$) was instilled twice during the visit. Good myosis resulted, with M.=$\frac{1}{10}$, V.=$\frac{16}{XX}$ (dark day).

January 12.—Several instillations of pilocarpine hydrochlorate ($\frac{1}{120}$), made during the visit, raised M. to $\frac{1}{18}$: a subsequent instillation of eserine sulphate (gr.$\frac{1}{240}$) gave M.=$\frac{1}{10}$, with somewhat less contraction of the pupil than had been noted after the instillations made on previous days.

January 13, 1 P. M.—Instillation of pilocarpine hydrochlorate

(gr.$\frac{1}{240}$) three times before 8 A. M., but not later: V.=$\frac{16}{\text{lxiv}}$, raised by— $\frac{1}{30}$ to $\frac{16}{\text{xx}}$. No myotic was used at this visit.

January 14.—Solution of pilocarpine hydrochlorate (gr.$\frac{1}{240}$) had been instilled the night before and early in the morning, three hours before the visit: diameter of pupil=6 mm; M.=$\frac{1}{27}$, V.=$\frac{16}{\text{xxv}}$.

Instillation of eserine sulphate (gr.$\frac{1}{240}$) contracted the pupil after one hour to 3 mm.

With —$\frac{1}{24}$ V.=$\frac{16}{\text{cxxvi}}$,
" —$\frac{1}{16}$ V.=$\frac{16}{\text{lxiv}}$,
" —$\frac{1}{14}$ V.=$\frac{16}{\text{xl}}$,
" —$\frac{1}{12}$ V.=$\frac{16}{\text{xxxii}}$,
" —$\frac{1}{11}$ V.=$\frac{16}{\text{xvi}}$,
" —$\frac{1}{10}$ V.=$\frac{16}{\text{xx}}$,
" —$\frac{1}{9}$ V.=$\frac{16}{\text{xxv}}$,
" —$\frac{1}{8}$ V.=$\frac{16}{\text{xl}}$.

Increase in R., after action of one instillation of eserine sulphate for one hour, =$\frac{1}{11}$ —$\frac{1}{27}$, =$\frac{1}{18}$.

An hour and a half after these measurements, the pupil having become noticeably larger, M. =$\frac{1}{18}$, V.=$\frac{16}{\text{xvi}}$.

In this case I am certain that the extent of the rupture of the zonula was nearly or quite equal to one-half of its entire circumference, and that, therefore, there could have been no possibility of effective traction upon the lens capsule through the zonular fibres.

The refraction, if assumed to have been originally the same as in the fellow eye, (Hm. =.5 d.) had undergone an increase of about 2. d.; namely, to M. =1.5 d.

Under extreme changes of illumination, and during strong convergence, with full accommodation in the fellow eye, no contraction of the pupil was observed, nor could any accommodative change be detected by carefully directed tests made with the visual axes parallel and under different degrees of convergence for the near.

The instillation of a solution of eserine sulphate(gr.$\frac{1}{240}$)was always followed, although somewhat slowly, by contraction of the pupil (usually from 6 mm. to 3 mm. diameter), and by an increase in

the refraction of from 1.5 d. to 2. d. A similar contraction of the pupil and increase in the refraction were evoked by instillations of pilocarpine hydrochlorate, but more slowly than by eserine, and generally in a somewhat lesser degree.

It seems difficult to avoid the conclusion that in this case the lens, largely detached from its suspensory ligament and insensible to the ordinary stimuli to accommodative change in convexity, had nevertheless retained the power of changing its form under the extraordinary local stimulus of the instillation of a myotic solution. If this conclusion is accepted, it would seem to point to an active contraction of the lens fibres as the essential act in accommodation, thus recalling the views of John Hunter (who erroneously supposed that he had demonstrated muscular fibres in the lens), and of Thomas Young, (who asserted and fairly proved the fact of the increased convexity of the lens in accommodation).

A CASE OF CORECTOPIA.

BY G. M. SMITH, M. D., BLOOMINGTON.

Among the congenital anomalies of the iris there is none, perhaps, so rare as corectopia. The following case (observed in Miss S., æt. 25), came under my notice March 26.

The eccentric displacement of the pupils was very marked and the same in both eyes. The pupils (see Figure 6[1]) were situated near the temporal part of the corneo-scleral margin, from which they were separated only by a very narrow strip of iris-tissue.

The anterior chamber was very deep, and the irides being tremulous, I suspected at first that aphakia was connected with the corectopia. Yet, the fact that there was a range of accommodation of eight inches, established, I think, the presence

1 The pupil lies a little too high in the drawing.

of a crystalline lens. The eyes were hypermetropic 6 D. I had glasses ground with the centre to correspond with the pupils, which were two and three-fourth inches apart.

This correction of the defect of the refractive power was highly appreciated by the lady. She was enabled to read quite

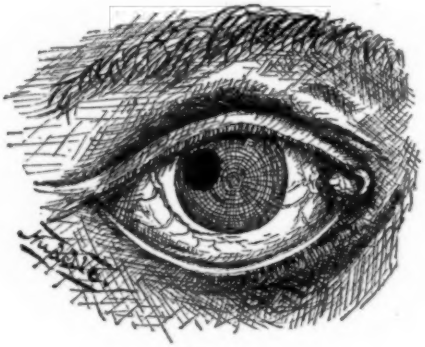

FIG. 6.

easily with both eyes at the same time (? EDITOR), having before been unable to use both eyes together.

It was my wish to make a thorough examination of her eyes, and especially with regard to the condition of the lens, but she would not consent to the use of atropia.

ATROPHY OF THE OPTIC NERVE. MULTIPLE SCLEROSIS OR SPASTIC PARALYSIS. DIFFERENTIAL DIAGNOSIS.

BY DR. H. W. HERMANN, ST. LOUIS, MO.

Atrophy of the optic nerve has often been found in diseases of the spinal cord. It was first and most frequently observed in connection with tabes dorsualis. But other spinal affections

must also be charged with a share in producing this dire complication. It has been reported in a small number of cases of multiple sclerosis. The following case I consider as belonging to the latter class, although it might be taken to be a case of spastic paralysis.

Mr. W., æt. 51, formerly a saloon-keeper, was well until seven years ago, when he suffered from gastritis caused by excessive drinking. Four years ago he had constant headache and occasionally a high temperature, attacks of dizziness and "crises cardialgiques."

Sight began to fail at about this time. Subsequently he had superficial gangrene of the toes.

On May 27. 1881, (examination by Dr. Alt), he presented myosis, atrophy of both optic nerves. V$\frac{20}{200}$. Visual field concentrically narrowed in both eyes. Color perception normal.

On August 3. 1882, when he came to me for examination, his condition was as follows: Well nourished man, face florid, perspires easily, except over lower extremities, digestion fair, irritability of bladder, mentally slow, speech heavy though not scanning, occasional headache, pupils react on light, myosis.

Tendon reflexes increased, also skin reflexes. Sudden noise also excites reflexes. Formication and pain in extremities, lower extremities feel cold and numb. Sensibility to touch diminished in legs. Left leg is slightly paretic. Can only walk badly without a cane. Some muscular rigidity. Right planta painful on pressure in some spots. Feet become livid after standing a while. Skin over little toe of right foot thin; on second toe signs of beginning gangrene.

In May following, gangrene of the skin of the great toe of the left foot took place which granulated over in six weeks, and was exquisitely painful, causing sleeplessness and occasional delirium. In March, 1884, two other toes were affected in a similar way.

March 23. 1884.—Dr. Alt again examined the eyes and found myosis; pupils contract, however, slightly on strong light as well as on accommodative effort. V=$\frac{20}{200}$. Field the same as three years ago. Atrophy of the optic nerves also the same.

Reads some letters of Jaeger 15. Color-sense normal. Slight paresis of the rectus internus of right eye which causes him to reach beyond objects on left side. The other symptoms are nearly the same as above described. Tremor is not very noticeable. The mental impairment is increased. Slight ataxia; tendon reflexes increased.

In this case the atrophy constitutes a very prominent symptom ; but from its appearance alone we could not make the differential diagnosis between the various organic diseases which may produce it. Only when taking the degree of functional disturbance, the history, and the other symptoms in consideration, can we make an exact diagnosis.

The muscular rigidity and increased tendon reflexes point to spastic paralysis, (sclerosis of the anterio-lateral columns). But these symptoms often occur also in multiple sclerosis and are only slightly developed in this case. Morever, atrophy of the optic nerve has never been observed in spastic paralysis. As a further means of distinction we have the bladder symptoms, the mental degeneration, anesthesia and the gangrenous patches on the toes.

We may consider the alcoholic excesses to be an etiological moment in the production of the sclerosis, as we know that chronic alcoholism gives rise to arterial degeneration, though this by itself would not suffice to explain all the symptoms in this case. Why atrophy of the optic nerve should occur in spinal affections is a mooted question as we know of no direct connection between the optic nerve and the spinal cord.

The cilio-spinal centre, situated in the heighth of the last cervical and beginning of the first dorsal vertebræ, communicates indirectly by means of the sympathetic nerve with the iris; but no direct connection has yet been proven for it with the optic nerve.

In affections of the meninges the changes in the optic disk may be easily explained by the contiguous inflammation of the optic sheaths or by an increase of the cerebro-spinal fluid which then mechanically interferes with the circulation of the retinal vessels, as has been proven experimentally by M. W. af Schul-

ten,[1] and others. In these cases the atrophy is secondary to the circulatory complication.

When any brain symptoms are manifest in multiple sclerosis, it is convenient to assume a patch of sclerosis in the optic nerve or tract, but this is not always found to be the case, and we must hope that future developments will disclose to us the paths of this connection between the optic nerve and spinal cord which evidently exists.

NOTES ON STRABISMUS.

DR. S. C. AYRES, CINCINNATI.

The development of convergent strabismus independent of paralysis or paresis of the abductors in the adult is a matter of rare occurrence. We are all familiar with strabismus as it is generally seen in children. We form our own opinions as to its causes independent of the so-called cause assigned by the parents. Such cases are usually tested to ascertain the refraction, or it is estimated with the ophthalmoscope if the child is young. The tenotomy of one or both muscles is made and proper correcting glasses are ordered, if they are required, and we have done all that is indicated. The majority of all cases of squint develop from the second to the seventh year although there are a few where it is congenital or develops within a few days after birth. The theory that hypermetropia is the cause of squint has for years been generally adopted, but it is not entirely satisfactory in this that it does not explain why many more hyperopes do not suffer from squint.

The importance of anomalies of refraction cannot be denied and yet we must conclude that the value of anomalies of the *muscles* has been underestimated. Prof. H. D. Noyes in his work on "Diseases of the Eye," says that Donders regarded "converging squint and hypermetropia as standing almost universally for cause and effect." But he adds "large observation has

1. Experimentela och kliniska undersœkingar betr. hiærnskador och deras inflytande pa œgats cirkulationsfœrhallanden. Helsingfors.

modified these views, and while we find that hyperopia acts the most important part in the production of converging squint, we have many statistics to show that essential muscular defects are also operative." Prof. Schweigger has antagonized the generally adopted theory of the importance of *H* and gives extensive statistics to show the importance of insufficiency of the external recti, or as he terms it, elastic preponderance of the internal recti muscles in producing strabismus. It is in the experience of every observer that the highest degrees of *H* are not most frequently associated with squint, but on the contrary the moderate degrees. We also frequently see cases of amblyopia congenita where there is no squint and where the good eye has a slight or even a high degree of hyperopia. In the following case there was a high degree of H (8 D.) and one eye was very amblyopic and yet squint did not develop until the twentieth year.

Case I.—Miss S. S. æt. 20. Complained of asthenopic symptoms and was tested under duboisia and found to have $H=8$ *D* R. E. V.$=04$. The left eye was very amblyopic but was not convergent. She was ordered $+4.5$ *D* for constant use. About six months afterwards (in February, 1881), she returned with the report that she could not use her glasses, and that for two or three weeks she had been seeing double. The diplopia was so annoying that she could not attend to her business, which was that of a saleswoman. Examination showed that there was a slight convergence of the left eye. She stated that it had developed gradually and was now a fixed condition, and that she could no longer retain her position in consequence of the annoyance of the double vision. The visual acuity of the right eye was found to be about as it was when previously examined. There was no known cause for the development of the strabismus as she had received no injury of the head, and there was no paresis of the left abductors. She was kept under observation about a month and then a tenotomy of the left internus was made. The diplopia was at once relieved and she was ordered $+5$. *D* for constant use. It is now three years since the operation was done and there has been no difficulty in using her eyes. She wears her glasses constantly.

CASE II.—Development of strabismus with annoying diplopia. J. D. B., æt. 29, has convergent strabismus with a most annoying diplopia which prevents him from attending to his business. These conditions developed about six years ago. At first the diplopia was not constant, but gradually it became a fixed condition and now renders his life very uncomfortable. He can neither blend the two images nor ignore one of them. There is permanent contraction of the right internal rectus muscle and the only chance for relief is in a tenotomy. This was done by Dr. E. Williams with very satisfactory results. The diplopia was relieved and he resumed his business and was enabled to use his eyes comfortably.

The visual acuity of the eyes was not noted.

CASE III.—Traumatic strabismus—caused by an operation for Dacryocystitis.

Miss C. æt. 26.—Applied for treatment in consequence of a very annoying diplopia from which she had been suffering for about three months. She gave the following interesting and unique history.

Three months ago she submitted to an operation for dacryocystitis of the left side. It was extremely painful and the operator was a long time in doing it, and judging from what she said, it was done in a bungling manner. The tissues in and around the inner canthus were very much bruised and considerble swelling and tumefaction followed.

She says she saw double immediately after the operation. This did not subside with the local swelling but persisted up to the time of examination. The images were harmonious and it was impossible for her to blend them. There was a marked contraction of the internal rectus of the left eye, but vision was perfect, as was that of the right eye. A tenotomy of the left internus was proposed to which she readily consented. The tendinous insertion of the muscle in the sclerotic was severed close to the sclerotic, care being taken not to incise the capsule of Tenon more than was absolutely necessary. The diplopia was immediately relieved and she shortly returned home. Two years and a half have now passed since the operation, and so far there has been no return of the trouble.

CASE IV.—Strabismus following an abscess of the upper lid. Mr. A., æt. 37, suffered from an abscess of the upper right upper lid, about four months ago. There was marked swelling and œdema of the upper lid and for a while the eye could not be opened. Poultices were applied and in time the abscess was opened. The swelling gradually subsided and the lid resumed its normal appearance. In consequence of the inflammation he kept the eye bandaged for several days. When he removed the bandage he found that he saw everything double. He tried in vain to walk around and attend to business with both eyes uncovered and was finally compelled to wear a patch of black silk over the right eye. He waited, hoping that the visual disturbance would disappear in time, but as it did not he applied for relief. The eyes tested separately were about equal and there was no fundamental lesion. There was a permanent contraction of the right internal rectus and he was advised to submit to a tenotomy of the muscle as the only means of relief. The operation was done in the ordinary way and the results were as immediate as they were satisfactory. The diplopia was relieved and he was restored at once to binocular vision.

TWO CASES OF DOUBLE, CONGENITAL, SYMMETRICAL ECTOPIA LENTIS IN SISTERS.

BY B. E. FRYER, M. D., U. S. ARMY, FORT LEAVENWORTH, KANS.

Cases of that form of incompleteness or irregularity of development of the eye, ectopia lentis, though not uncommon, are sufficiently rare when occuring in two children of the same family as to merit reporting.

CASE I.—Josephine Vogel, aged seven years (born in Nashville, Tenn., March 24. 1875), was brought to my office, August 27. 1882 by her father. The child was rather small for her years, but seemed well nourished and quite intelligent. Her vision in both eyes was $\frac{20}{200}$. On dilating the pupils it was found that ectopia lentis existed in both eyes, the lenses being directly outwards towards the temples, the extent of the malposition, as

compared with the natural site, was more than half the diameter of the lens. This position was similar in both eyes, and both lenses were alike in not being free from a slight cloudiness. With +6 the child was able to read the short words in No. 3 of Snellen at eight inches without much difficulty. The eyes were of normal size.

On describing the condition to her father he said his younger child also had difficulty in vision, and on September 3. 1882, he brought her to my office.

Case II.—Wilhelmina Vogel, born in Nashville, Tenn., September 16. 1877, therefore nearly five years of age. The child was delicate and small for its years. The pupils being dilated with atropia I found that the lenses in both eyes were placed symmetrically upwards and outwards about 45°, the extent of the malposition being one-half the diameter of the lens. The child being but five years old no satisfactory examination of visual acuity could be made. Gave +8 glasses.

Both of these cases have since been lost sight of.

DOUBLE CONGENITAL ZONULAR CATARACT IN AN INFANT FOUR MONTHS OLD, IN WHICH BOTH ATROPIA AND DUBOISIA PRODUCED BUT SLIGHT PUPILLARY DILATATION.

BY B. E. FRYER, M. D., U. S. ARMY, FORT LEAVENWORTH, KANS.

Mr. and Mrs. A. Gay, of Concordia, Kansas, brought their baby to my office, February 25. 1884, in reference to its eyes. The child was a healthy looking boy of four months of age, well-nourished and developed, and in good health. Both parents healthy, the father especially so. No specific taint on either side.

The child's eyes were of normal size; slight convergence of axes existed, and occasionally nystagmic movement of the eyeballs. The irides, which were bluish gray, appeared perfectly natural and healthy, and though the pupils were somewhat small they responded to the effect of light. Both lenses had zonular

cataract in them, with a slight cloudiness beyond the opaque zone.

The day the child was brought to me I pe cilled the conjunctiva of the lower lid with a freshly made solution of atropine (one grain to one ounce of water) several times, and also dropped on the everted lower lid a drop or two, and again repeated the pencillings making several applications in the morning from 9 till 2. In about half an hour from the first application of the atropia there was a dilatation of the pupil not amounting to more than two and a half millimetres. This was a little over a millimetre of dilatation. The application of the atropine was continued on the following and also on the third day, but with no greater effect. The atropine solution was a freshly made one, and one I was using in other cases with maximum effect upon the pupil.

I determined to try duboisia on the fourth day, and had a solution made of that alkaloid (one grain to one ounce) from some of Merk's manufacture. I used this as I had the atropia and continued it three days, but no further dilatation of the pupils was obtained than that had on the first day's use of the atropia. On this I directed the parents to take the child home and to pencil the conjunctiva of the lower lid each day with the atropia solution and to bring the child to me again in one month.

On April 12. last, the child was brought again to my office and the mother stated that there had been no greater enlargement of the pupil during the interval than before, and for a few days no application of the drug had been made, and its effects at this time had completely subsided. I now had an opportunity of again examining the child's pupils free from atropine, and also while it was asleep, during which latter the pupils were contracted to a small pin's head in diameter, enlarging to nearly a millimetre and a half on waking. I now again began the use of the atropia solution in the same way as before, instilling drops more frequently perhaps. I continued this two days. The result was the same as at first, viz.: A pupillary expansion of a trifling over two and a half millimetres. I then resorted to the duboisia again for the same time, but with no further improvement in the size of the pupils, though the mydriatics had been pushed to the production of a constitutional effect.

The enlargement of the pupils slight as it was, was equal in extent in both eyes, and was symetrical and both pupils were circular at all times.

That there were no synechiæ to interfere with the pupillary expansion would appear from the complete pupillary contraction during sleep; from the circular form of the pupils, both when contracted and also when under the effect of the mydriatics; and from the fact that there was no evidence of any iritic inflammation and no sign that it had occurred, the irides appearing perfectly normal in every respect. Examination was made by oblique light and with the ophthalmoscope.

The child's perception of light was apparently moderately good, the eyes following the movement of bright objects and closing the lids on the approach of opaque bodies to the eyes.

The case is reported with the hope of bringing to record similar ones; the condition being of interest, presenting as it does an element of difficulty in any operative procedure adopted for the removal of the obstruction of the passage of light to the retina. I cannot offer any satisfactory theory to account for the cause of the condition which probably ultimately depended on a rigidity of the musular fibres of the sphincter pupillæ, which neither of the mydriatics used were sufficient to overcome.

A CASE OF SYMPATHETIC NEURO-RETINITIS. REMARKS ON SYMPATHETIC OPHTHALMIA.

BY ADOLF ALT, M. D.

[Continued from No. 1. page 32.]

I think the following supposition will explain in the simplest manner how these conditions could come about: An inflammatory process in the angle of the iris having given rise to the formation of new connective tissue, this gradually grew forwards between the fibres of the ligamentum pectinatum, and thus split *Descemet's* membrane in two halves.

A small fold of iris-tissue was attached to the hyaline membrane lining the newly formed tissue, and was considerably stretched. The iris-tissue as a whole was filled with hymphoid

cells, many of which contained a number of pigment molecules. A great deal of pigment was lying free in the tissue, while the stellated pigmented parenchyma-cells of the iris were wanting almost altogether. The few blood-vessels which could be seen in the iris were hyperæmic.

The tissue of the ciliary body was largely infiltrated with round-cells in its anterior portion. On the inner surface of the ciliary body some of the torn fibres of the suspensory ligament of the lens were visible. The fibres of the ciliary muscle ran nearly all in a longitudinal direction. The blood-vessels were hyperæmic.

In spite of the dislocation of the lens and in spite of the obliteration by the newly formed tissue of the communication between the anterior chamber and the drainage system in the scleral tissue no glancomatous symptoms had ever been observed in this case. This was surely not due to the iridectomy.

The ciliary nerves which I examined with especial care showed no pathological changes.

The choroid was normal and there was no posterior scleral staphyloma.

The optic nerve was destroyed by hypertrophy of its connective tissue and atrophy of the nervous elements. There was also diffuse atrophic retinitis. Most of the retinal blood-vessels were obliterated; of the remaining ones some were empty, some hyperæmic. The internal limiting membrane of the retina was detached from the latter in a number of places by a gelatinous exudation.

The lens-capsule was intact; some torn fibres of the suspensory ligament were attached to its equatorial parts. The capsule was wrinkled, the lens shrunken. The pyramidal cataract lay near, but not exactly in the anterior pole of the lens. It was irregularly shaped, not cone-like as usual, but rounded off and thicker at its apex than at its base. It consisted of connective tissue arranged in lamellæ void of cellular elements. Near the base this tissue contained a large quantity of lime, as did the anterior part of the lens. The capsular epithelium had disappeared. The remainder of the lens was in a state of regressive metamorphosis.

The fluid vitreous body contained a number of flocks consisting of fibrine and young connective tissue.

Remarks. The remarks I intended to make in connection with this case have quite unexpectedly received another direction than the one I had in mind.

In the March number of the Archives of Ophthalmology Dr. Theobald, of Baltimore, has done me the favor of kindly reviewing my statistics on sympathetic ophthalmia (reported in 1876 in the same archives) in a paper he entitles: *Some recent theories regarding the pathogeny of sympathetic ophthalmia, viewed from a macroscopic standpoint.* I have never taken up a literary fight, nor do I intend to now; yet the manner in which Dr. Theobald expresses himself is such that I cannot remain silent, limiting myself, however, to the following statements which will cover all I shall have to say in this matter. I shall simply correct my own mistakes and what is wrongly reported, or willingly or unwillingly distorted by Dr. Theobald, in order to prove his subject. Thus I hope to help in saving the advance in strict knowledge which has been made within the last decade in regard to the pathogeny of sympathetic ophthalmia from being lost again through statements from a "macroscopical standpoint," which come from a gentleman who would think it as much of an offense to have his veracity and earnestness in search after scientific truth doubted, as I do.

His first statement is that my statistics actually refer to 112, not 110 eyes, as I stated. This point might appear well taken, yet I did not include in the statistics two eyes, which had been removed for sympathetic keratitis, as I considered them (see page 475) doubtful, to say the least.

In my tabular statement the ciliary nerves are mentioned in 45 cases, not as I had erroneously stated, in 43. In 7 of these cases they were pressed upon by exudation or entered into the tissue of the scar, without any apparent change of their nervous elements.[1] What right Dr. Theobald has to count these as cases in

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1. In the translation of my paper, which I had written in German, an error occurred, which I did not notice at the time. In the case of *A. Prichard*, table I, fifth page, a ciliary nerve is mentioned as being *much torn*, this ought to read: one *considerably stretched* or *dragged upon* (gezerrt).

which the ciliary nerves showed pathological changes I cannot perceive. In 6 cases only there were distinct histological changes visible in the tissue of the ciliary nerves and in 1 case they were "not discoverable," that is they had been destroyed in the inflammatory process and thus had surely undergone pathological changes. This means that the tissue of the ciliary nerves was found to be, or to have been, diseased in 7 cases of the 45, or in 15.5 per cent. Or, take the 112 eyes, then the ciliary nerves have been mentioned 46 times and been found pathologically changed 8 times = 17.3 per cent. and not as he will have his readers believe in 32.6 per cent. being very careful to state, that he did not think it necessary to carry the fractions beyond the first decimal. The "gross pathological changes" do not belong here.

In my tabular statement the conditions of the optic nerve and retina are mentioned in 85 cases, and that would mean, as their *normal* condition is never mentioned, that in 100 per cent. of the cases in which their condition is stated this was pathological. Thinking this wrong[1] I calculated the percentage, as if the condition had been stated in all eyes, which would make 77.2 per cent. or, for 112 eyes, in which the optic nerve and retina were mentioned as pathologically changed 86 times, in 76.7 per cent. Optic nerve and retina are one organ, as much so as choroid, ciliary body and iris are one organ: the uveal tract. No change in the one part can exist long without affecting the other part of one of these organs. Therefore, I most logically counted the affections of the optic nerve and retina (optic nerve, or retina, or both) as the affections of one organ. If Dr. Theobald had examined many such eyes with detachment of the retina, he would know that this "gross change" involves also histological changes in almost every case. As he evidently has not, it is not strange that through his spectacles my statements should appear as "*most grossly misinterpreted pathologico-anatomical observations.*"

But now to the main point. Dr. Theobald honors me by giv-

1 I have no doubt that retina and optic nerve were examined in every case, although this is not stated, while I seriously doubt that the ciliary nerves were examined especially in all the cases I had found in the literature on this subject.

ing me credit for having initiated a new movement in the theory of the pathogeny of sympathetic ophthalmia. Be it so or not—surely the theory of transmission of the inflammation from one eye to its fellow by means of the optic nerve alone, was not the conclusion I came to in the paper against which he militates. The conclusion there (see page 475) is, "*That the entire nervous apparatus of the diseased eye participates in the transmission of the affection to the other.*" Moreover, in a paper written in 1878, (which Dr. Theobald knows, as he refers to it) on the condition of the ciliary nerves in an eye suffering from purulent panophthalmitis, enucleated on account of sympathetic iritis, I stated again, "*that this is* ONE *of the ways in which sympathetic affections may be brought about, i. e. by direct transmission of the inflammation*, scil. by way of the optic nerve.

I cannot help feeling honored by the remarks of Dr. Theobald, although they were intended quite differently. Yet the honor is really due to the gentlemen who, since my paper appeared, have found themselves in the position to claim that the optic nerve and its sheaths alone transmit the inflammation from one eye to the other in sympathetic ophthalmia, while sympathetic irritation is simply a reflex-neurosis. I here refer especially to *Knies, Leber and Deutschmann.*

In thus defending the statements made in my paper in 1876, I do not wish it to be understood that I am to-day of the same opinion as at that time. On the contrary, I intend to state, that my position in this question to-day is the one expressed by *Knies* in his paper on sympathetic ophthalmia in "Beitraege zur Ophthalmologie als Festgabe, Friedrich Horner, etc., gewidmet. Wiesbaden, 1881," which Dr. Theobald has evidently no knowledge of. I do, however, not consider sympathetic ophthalmia to be simply "a sero-plastic uveitis," as my friend *Knies* does. With this exception, *Knies* says there: "*The symptoms of so-called sympathetic irritation are reflex-neuroses in the sphere of the motor, sensorial and vasomotor functions and by way of the ciliary nerves.*" Further on he states: "*The sympathetic inflammation is an inflammation (sero-plastic uveitis) which travels from one eye to the other along the optic nerve and its pia mater sheath by way of the chiasma.*

Of course, the direct anatomical proof is as yet wanting for this theory, because, if it was not wanting the question would be no longer subject to theories. Yet, the theory of transmission by the ciliary nerves has only an extremely small anatomical support, while excellent clinicists and microscopists have many times tried to find direct proofs of this theory and have failed to do so to their own dismay. It has appeared to me just as strange that Dr. W. C. Ayres was able to find pathological changes in the ciliary nerves in every one of his seven cases (Arch. of Ophth. Vol. X), as it is to Dr. Theobald that I found these nerves normal in fourteen out of fifteen cases which I examined and in which the eyes had caused sympathetic irritation. (By using his own method of thought he ought here to have said, that I found the ciliary nerves normal in twelve out of fourteen cases in which I mentioned their conditions. As he wants to show my inaccuracies, he ought to be more accurate himself).

It is impossible to refute his further assertion "to the pathologists of the Cohnheim school" that reflex-congestion will lead to inflammatory changes, in any other way here, but by stating my equally firmly rooted conviction that reflex-congestion never yet has caused inflammation. On the other hand, when Dr. Theobald (page 80), in order to prove an influence of "trophic" nerve-fibres upon the production of sympathetic inflammation, cites the ulcerative keratitis, which (though with reserve) is called neuro-paralytic, occurring after division of the trigeminus nerve, he forgets that such a keratitis would be primarily a *necrosis* and not an active inflammatory process, in other words he does not prove anything by this simile.

Being greatly interested in this, one of the most important, subjects of ophthalmology, I have given it special time and observation, and, induced by a remark made by Dr. Williams, of Cincinnati, at the meeting of the International Congress of Ophthalmology, (see report page 45), in 1876, who stated that he found active neuro-retinitis in two eyes he enucleated after serious injuries, I carefully examined a number of injured eyes with the ophthalmoscope as soon as possible after they had been injured; and I find by referring to my books, that in five cases of severe injuries in which an ophthalmoscopic examination was possible there

existed, in periods varying from a few days to three weeks after the injury, a decidedly well-marked neuro-retinitis with tortuousness of the retinal veins.

The fact that we usually *see* the first signs of sympathetic ophthalmia appear in the anterior portion of the uveal tract is, therefore, no proof against the theory of direct transmission of such a traumatic neuro-retinitis from one eye to the other, because we do not, as a rule, see the cases early enough in order to diagnosticate such a possibly existing neuro-retinitis in the sympathetically affected eye. That some observers have seen similar conditions is, however, proven by the statement of *Dr. H. D. Noyes* (see page 47 report of the International Ophthalmologic Congress in 1876) who says: "*I have been accustomed * * * to lay great stress upon the peculiar delicate infiltration and haziness of the optic nerve of the eye*" (meaning the in eye danger from sympathy). As the first affected eye usually shows an affection of the uveal tract, such inflammation may be, and probably usually is, transmitted at the same time as the neuritis, by means of the optic nerve sheath (see my paper on sympathetic neuro-retinitis, the same report page 37).

Yet, after all, it will be impossible to convince an antagonist of being wrong by theorizing merely, nor is anything to be gained, until the microscope (not the macroscopic inspection) has revealed the real condition of things. *Deutschmann*, therefore, has made a number of experiments with a view to produce sympathetic ophthalmia. He has been (see Graefe's Archiv. xxviii, page 291) thus far successful in producing "sympathetic papillitis" and has demonstrated it anatomically. His paper also does not seem to have come to Dr. Theobald's knowledge and I take great pleasure in inviting his attention to it.

Experimenting in the same way on rabbits I have after numerous failures succeeded in producing a severe traumatic inflammation in one eye, which on the third day was followed by a well-marked iritis in the fellow. I had the honor to demonstrate this rabbit before the society of German physicians of St. Louis (Verein Deutcher Aerzte). If, as I think, since rabbits are, as far as I know, not especially apt to suffer from idiopathic iritis, this was actually a "sympathetic" iritis, the question will

be brought nearer a settlement by an examination of the eyes and nerves taken from this rabbit. On this I shall report in the next number of this journal.

[TO BE CONTINUED.]

CORRESPONDENCE.

The following letter was received from Dr. S. M. Burnett, in Washington, D. C.

* * * In reply to the communication of Dr. Coggin, in the last number of the AMERICAN JOURNAL OF OPHTHALMOLOGY, I would say, that I prepared three years ago for the National Board of Health, a nomenclature of ophthalmology and otology, which was published in a supplement to their Bulletin.

I did not know but that you might wish to republish this (with revisions) in your journal. If so, I will send you a revised copy for examination.

[The editor has asked Dr. Burnett to kindly forward this revised nomenclature, and hopes to be able to republish it in the next number of this journal.]

CORRIGENDA.—NO. 2.

Page 35, the 8th, 9th, and 10th lines should be the 1st, 2nd, and 3d.

Page 52, 20th line from below read V = 0.4, instead of V = 04.

Page 53, 11th line from below read *homonymous* instead of *harmonious.*

Page 54, 2d line from above read *right* instead of *upper right.*

Page 57, 1st line from below read *l*ymphoid instead of *h*ymphoid.

Page 58, 16th line from above read gla*u*comatous instead of gla*n*comatous.